U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 23 May 2007

CASE No.: 2001-BLA-701

In the Matter of:

F.W.,
Claimant

v.

ELKAY MINING COMPANY,
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest

Appearances:

Robert Cohen, Esquire
For Claimant

Kathy L. Snyder, Esquire
For Employer

BEFORE: MICHAEL P. LESNIAK
Administrative Law Judge

DECISION AND ORDER – AWARDING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq* (the Act). Regulations implementing the Act have been published by the Secretary of Labor in Title 20 of the Code of Regulations.

The Act provides benefits to persons totally disabled due to pneumoconiosis and to certain survivors of persons who had pneumoconiosis and were totally disabled at the time of their death or whose death was caused by pneumoconiosis. Pneumoconiosis is a chronic dust disease of the lungs, including respiratory and pulmonary impairments arising out of coal mine employment, and is commonly referred to as black lung or CWP.

A hearing was held before me on January 11, 2006 in Morgantown, West Virginia (see "Background and Procedural History", infra, for a complete account of the procedural history of this case). [1]

The findings of fact and conclusions of law that follow are based upon my analysis of the entire record, including all documentary evidence admitted, arguments made, and the testimony presented. Where pertinent, I have made credibility determinations concerning the evidence.

ISSUES

At the hearing the following issues remained contested:

(1) Whether the claim was timely filed;
(2) Whether Claimant has pneumoconiosis as defined by the Act and regulations;
(3) Whether his pneumoconiosis arose out of coal mine employment;
(4) Whether Claimant is totally disabled by a pulmonary impairment; and
(5) Whether his total disability is due to pneumoconiosis. (TR 11-12).

Although Employer contested the foregoing issues at the hearing, in its closing brief, counsel for Employer conceded the existence of pneumoconiosis based on the biopsy evidence and that Claimant had a totally disabling pulmonary impairment based on recent pulmonary function studies. (Employer's closing brief, page 10). I agree with Employer and find that the evidence of record establishes the presence of pneumoconiosis and the presence of a totally disabling pulmonary impairment. Therefore the sole issue to be decided is whether coal workers' pneumoconiosis caused or contributed to Claimant's pulmonary disability.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Background and Procedural History[2]

This case has a long and convoluted procedural history. Claimant filed his claim for benefits on June 8, 2000. (DX 1; DX 49). On October 31, 2000, the district director denied benefits. (DX 22). After the submission of additional evidence, the district director then issued a Proposed Decision and Order Awarding Benefits on November 20, 2000. (DX 32; DX 33). Employer disagreed and requested a formal hearing. (DX 46). The case was subsequently referred to the Office of Administrative Law Judges on April 15, 2001. (DX 48). After several continuances, the case was finally set for hearing on November 21, 2002 by Judge Fletcher E. Campbell, Jr. In the meanwhile, Claimant served interrogatories and requests for production of documents. Employer objected to Claimant's interrogatories as irrelevant, overly broad, and

[1] The following references will be used herein: TR for transcript, CX for Claimant's exhibit, DX for Director's exhibit, and EX for Employer/Carrier's exhibit.

[2] Given the filing date of this claim, subsequent to the effective date of the permanent criteria of Part 718, (i.e. March 31, 1980), the regulations set forth at 20 C.F.R. Part 718 will govern its adjudication. Because Claimant's last exposure to coal mine dust occurred in West Virginia this claim arises within the territorial jurisdiction of the United States Court of Appeals for the Fourth Circuit. *See Broyles v. Director, OWCP*, 143 F.3d 1348, 21 B.L.R. 2-369 (10th Cir. 1998).

unduly burdensome and requested a protective order. On October 17, 2002, Claimant filed a Motion to Compel Discovery or, alternatively, to exclude the opinions of Drs. Bush, Naeye, Bellotte and Fino. On November 5, 2002, Judge Campbell issued an Order Granting Claimant's Motion to Compel Discovery.

Employer filed a Motion to Stay the Proceedings before the Office of Administrative Law Judges together with a Notice of Appeal of the discovery order to the Benefits Review Board ("the Board"). On November 15, 2002, Dr. Gregory Fino filed a Motion to Intervene before the Board. On November 25, 2002, the Board issued an Order granting Employer's interlocutory appeal and Dr. Fino's petition to intervene. On November 12, 2003, the Board found that because Judge Campbell had not set forth reasoning for his Order Granting Claimant's Motion to Compel Discovery, the order must be vacated and the case was remanded for further consideration.

On remand, the issues included Claimant's motion to compel, Employer's various objections, and Employer's motion for a protective order. While the case was pending on remand, Claimant sent interrogatories and requests for production of documents to Dr. Fino in his capacity as intervenor. Dr. Fino filed a Motion to Quash the Interrogatories together with a request to withdraw from the case as an intervenor and proceed as amicus curie. On January 23, 2004, Judge Campbell issued an Order allowing the withdrawal for Dr. Fino as intervenor and allowing his participation as amicus curiae. The parties and Dr. Fino filed briefs on the discovery issues and on March 15, 2004, Judge Campbell issued a 12- page Order Compelling Discovery.

On March 25, 2004, Employer filed a second Notice of Appeal and Motion to Stay with the Board. Claimant filed a Motion to Dismiss the Notice of Appeal. On April 8, 2004, the Board issued an Order dismissing Employer's appeal as interlocutory.

On April 14, 2004, Employer sent a letter to Judge Campbell explaining that in order to protect its right to appellate review of the protective order it would not produce the requested information and documents. However, due to Judge Campbell's impending retirement, the case was re-assigned to Judge Gerald Tierney. Judge Tierney set a hearing for October 27, 2004. In the meanwhile, Claimant filed a Motion for Sanctions for Employer's refusal to comply with the Discovery Order and Employer filed a Motion for Reconsideration of the Order to Compel. At the hearing, Judge Tierney indicated he would issue a ruling following the hearing regarding the discovery issues. However, due to his impending retirement, Judge Tierney notified the parties that he would not be making a ruling on these issues but that the case would be re-assigned to yet another judge.

The case was subsequently assigned to me and a hearing was set for January 11, 2006. On November 9, 2005, I issued an Order Granting Employer's Motion for Reconsideration and Granting Claimant's Motion to Compel Discovery in Part. Specifically, I vacated the Order to Compel with regards to interrogatories 1 through 4 but affirmed the Order to Compel with regards to interrogatories 5 through 24. On November 28, 2005, Employer submitted a response to Claimant's interrogatories 5 through 24 for *in camera* review and notified the parties that its filing of responses to Claimant's discovery in camera constituted a Motion for Reconsideration.

Claimant filed a Motion for Reconsideration of my ruling on November 9, 2005 with respect to interrogatories 1 through 4 and filed a response to Employer's second Motion for Reconsideration. On December 1, 2005, Claimant submitted a second set of Interrogatories and Requests for Production of Documents to Employer which mirrored the prior interrogatories but asked about Employer's relationships with Drs. Oesterling, Spagnolo, and Rosenberg. On December 2, 2005, I issued an Order Granting Claimant's Motion for Reconsideration with regards to Interrogatories 1 through 4 and denied Employer's Motion for Reconsideration with regards to Interrogatories 5 though 24. On December 9, 2005, Employer sent an x-ray in response to Interrogatories 1 through 4 together with responses to Interrogatories 5 through 24.

On December 13, 2005, Employer submitted its responses to Claimant's second set of discovery requests by basically reiterating its objections outlined in the first set of discovery requests. On January 4, 2006, Claimant filed a Motion to Compel with regard to the second set of Interrogatories. On January 5, 2006, Employer filed a response to the Motion to Compel and filed a Request for Protective Order. At the hearing on January 11, 2006, I overruled Employer's objections and granted Claimant's Motion to Compel Discovery with respect to the second set of Interrogatories. Employer filed its responses to said discovery requests on January 30, 2006.

During the post-hearing deposition of Dr. Spagnolo, counsel for Claimant indicated that the Office of Administrative Law Judges had provided him with a DVD disc containing decisions issued between 2002 and 2005. On June 21, 2006, Employer filed a motion asking that Claimant be compelled to produce the DVD disc and to disclose all communications between his office and the Office of Administrative Law Judges regarding the DVD. On June 28, 2006, Claimant filed his response to said Motion. On June 29, 2006, I held a conference call regarding Employer's motion. On July 10, 2006, I issued an Order requiring Claimant to produce the DVD disc obtained from the Office of Administrative Law Judges and all written communications regarding how that disc was obtained.

On June 30, 2006, Claimant filed Combined Requests for Admissions, Interrogatories, and Request for Production of Documents to Employer regarding Dr. Spagnolo. On July 28, 2006, Employer filed a Motion to Strike References to the DVD disc from the record. Employer also filed its own Combined Requests for Admissions, Interrogatories, and Request for Production of Documents to Claimant regarding Dr. Cohen and a Motion to Compel answers. A telephone conference call was held on August 28, 2006 to discuss the outstanding discovery issues. Counsel for Claimant did not oppose Employer's Motion to Strike references to the DVD disc. Both parties withdrew their discovery requests and agreed that the record should be closed. Claimant filed his closing brief on March 2, 2007 and Employer filed its closing brief on February 7, 2007.

Hearing on January 11, 2006[3]

As noted above, a hearing was held on January 11, 2006 in Morgantown, West Virginia. At the hearing Claimant testified that he was born on March 4, 1938 and was married to his wife, L.W. (TR 13). He worked off and on for different coal companies and steel companies from 1961 to 1971. (TR 16-18). In June of 1971, Claimant started working for Badger Coal Company. (TR 18-19). He worked there until September of 1982. (TR 19). He worked the last seven years as a roof bolter. (TR 19). He described the work as being dusty. (TR 19). He did not wear any type of respiratory protection. (TR 20). Claimant was laid off in 1982. After that he worked various odd jobs including a three-month stint tearing down a boiler where he could have been exposed to asbestos. (TR 21). Claimant stated that he had 13 ½ years of coal mine employment. (TR 21).

Claimant was awarded disability benefits in the 1990s for his breathing. (TR 22). Claimant stated that he started having breathing problems in 1990. (TR 23). He was treated at the VA Hospital for colon cancer in 1993. Claimant's cancer came back and spread to his left lung. It was detected by a biopsy performed in 1999 at Ruby Memorial Hospital. (TR 24). Claimant testified that he first was told by Dr. Graber following the lung biopsy in 1999 that he had black lung. (TR 25). Prior to that operation no doctor had told him he was totally disabled due to black lung disease. (TR 25).[4] Dr. Jain had been treating Claimant for his breathing problems every three to four months since 2000. (TR 25). Dr. Jain prescribed breathing medications for Claimant. (TR 26). Claimant smoked less than a pack of cigarettes per day from 1955 to 1999. He added that during this period he quit smoking at least five times and each time he quit was for several years. He estimated that he smoked about 25 years. (TR 27-28). He did not mention to Dr. Graber that he intermittently stopped smoking over the years. (TR 28).

Medical Evidence

Because Employer conceded the existence of pneumoconiosis and the presence of a totally disabling pulmonary impairment, I will only summarize that evidence that is relevant to the remaining issue of whether coal workers' pneumoconiosis caused or contributed to Claimant's pulmonary disability.

Dr. F.A. Scatteregia

The medical report of Dr. Scatteregia is dated July 25, 200 and appears at DX 29. He examined Claimant at the request of the Department of Labor. He noted a coal mine history of 11 years of underground coal mine employment. Claimant reported a family medical history of

[3] At the hearing, Director's exhibits 1 through 49 were admitted into evidence. (TR 4-5). In addition, Claimant's exhibits 1 through 14 and Employer's exhibits 1 through 46 were admitted into evidence. (TR 5-6). It should be noted that since Claimant filed his claim for benefits before the amended regulations were enacted (i.e. January 19, 2001) the limitations of evidence contained within 725.414 are not applicable to this matter.

[4] As noted above, Employer contested the issue of timeliness at the hearing. I find credible Claimant's testimony that he first learned of having black lung following his lung biopsy in 1999. Employer has not offered any evidence to the contrary. Accordingly, I find that Claimant timely filed his application for black lung benefits.

high blood pressure, heart disease, diabetes, cancer, asthma, and stroke. Claimant had a medical history of pneumonia, pleurisy, attacks of wheezing, chronic bronchitis, bronchial asthma, heart disease, allergies, and cancer of the lungs and colon. He noted a smoking history of one pack of cigarettes per day off and on for 25 years and ending in 1998. Claimant's chief complaints included sputum, wheezing, dyspnea, cough, chest pain, orthopnea, and paroxysmal nocturnal dyspnea. After reviewing a chest x-ray, pulmonary function studies, and arterial blood gases, Dr. Scatteregia diagnosed Claimant as having chronic bronchitis, pulmonary fibrosis, status post thoracotomy, and questionable coal workers' pneumoconiosis. He attributed these diagnoses to genetics, environment, cigarettes, and coal mine dust. He opined Claimant suffered from a moderate obstructive impairment which may have prevented Claimant from performing his last coal mine job. This impairment was caused primarily by chronic bronchitis and pulmonary fibrosis.

The deposition of Dr. Scattaregia was taken on April 6, 2001 and appears at EX 1. Counsel for Claimant did not appear at the deposition. Dr. Scattaregia is Board-eligible in Internal Medicine and conducted about 50 to 100 black lung evaluations each year. He noted a 30-pack year smoking history. Based on the information he had at the time of the examination he was not sure of the presence of pneumoconiosis. However, prior to the deposition he reviewed other medical evidence, primarily the medical report of Dr. Naeye. Based on his review of the pathology, Dr. Scattaregia stated that Claimant had pulmonary fibrosis, carcinoma, and evidence of very slight simple pneumoconiosis. He noted that the CWP was focal and not thought to have resulted in any measurable abnormality in lung function. He stated that Claimant's moderate vent obstruction was caused by smoker's bronchitis. He did not believe CWP contributed in any significant degree to said impairment. He added that he would defer to Dr. Bellotte's opinion based on his credentials and that he agreed with the opinion of Dr. Naeye.

Dr. D. Gaziano

On September 20, 2000, Dr. Gaziano responded to a request from the claims examiner to render an opinion on whether the miner's total disability from CWP was caused by his 8 ½ years of coal mine employment. (DX 29). He noted that 8 ½ years was not sufficient to explain his total disability and added that cigarette smoking, lung cancer surgery, COPD, and welding fumes all played important parts in Claimant's disability.

After an additional investigation into Claimant's coal mine employment history by the claims examiner it was determined Claimant had worked 13 ½ years in underground coal mines. On November 13, 2000, Dr. Gaziano stated, based on the new occupational history, that Claimant's impairment was due to a significant degree to his occupational exposure to coal mine dust in coal mine work. (DX 31).

The medical report of Dr. Gaziano is dated October 24, 2002 and appears at CX 6. Dr. Gaziano is Board-Certified in Internal Medicine and Pulmonary Disease. He conducted a medical record review at the request of Claimant. He noted that the amount of tissue removed for the biopsy was extremely limited and represented no more than 15% of the Claimant's total lung tissue. He agreed with Dr. Alan Ducatman's opinion that to base impairment on limited biopsy specimen was clearly wrong. He noted that Claimant's smoking and coal mine

employment contributed to his lung disease as well as emphysema. He concluded Claimant had clinical and pathological evidence of pneumoconiosis and the only clinical and pathological measurement of the degree of impairment is the pulmonary function test that indicated a severe and disabling degree of impairment. He added that the type of chemotherapy given to Claimant would not have produced Claimant's pulmonary impairment. He opined that Claimant's coal mine employment of sufficient duration, the presence of CWP, and pulmonary function studies verifying severe impairment equals disability caused or contributed to by underground coal mining.

Dr. John A. Bellotte

The medical report of Dr. Bellotte is dated January 23, 2001 and appears at DX 40. Dr. Bellotte is Board-Certified in Internal Medicine and Pulmonary Medicine and is a B-reader of chest x-rays. (DX 39). He examined Claimant at the request of Employer. He noted an occupational history of 13 years of coal mine employment with 11 years underground. He reviewed Claimant's family medical history and individual medical history. Claimant admitted to a smoking history of one pack of cigarettes per day for 20 years noting that he stopped smoking several times. Claimant's chief complaints were cough with sputum production, wheezing, right-sided chest pain, and occasional paroxysmal nocturnal dyspnea. Physical examination of the chest and lungs revealed wheezing. A pulmonary function study showed severe obstructive vent defect, the chest x-ray showed bullous emphysema and old granulomatous disease, and arterial blood gases showed mild hypoxemia. Dr. Bellotte reviewed and summarized some additional medical records. He concluded that there was insufficient evidence to justify a diagnosis of CWP. He admitted Claimant had a totally disabling pulmonary impairment related to his COPD, chronic bronchitis, and emphysema. He attributed these diagnoses partially to tobacco abuse and partially to remodeling secondary to his asthmatic condition. He noted that Claimant also had pulmonary fibrosis related to malignancy and chemotherapy. He stated that the total disability was not caused in whole or in part by coal workers' pneumoconiosis.

The deposition of Dr. Bellotte was taken on August 3, 2001 and appears at EX 7. Counsel for Claimant did not appear. Prior to the deposition he reviewed additional medical records including the medical reports of Drs. Fino and Bush. He noted that pathology showed a mild pneumoconiosis was present. He attributed the pleural fibrosis to an infectious disease such as pneumonia. He opined that Claimant's pulmonary impairment was due to tobacco abuse, asthma, colon cancer and subsequent treatment. He concluded that CWP played no role in causing Claimant's disability.

Dr. Gregory Fino

The medical report of Dr. Fino is dated July 16, 2001 and appears at EX 5. Dr. Fino is Board-Certified in Internal Medicine and Pulmonary Disease. (EX 6). Dr. Fino conducted a medical record review at the request of Employer. He noted an occupational history of 11 years of coal mine employment. He reviewed the pathology reports of Drs. Naeye and Bush and the medical report of Dr. Bellotte. He noted a smoking history ranging from 20 to 40 pack years. Dr. Fino stated that Claimant had a very severe vent abnormality but that the pathological

findings showed only a very mild pneumoconiosis. He added that the chest x-rays and CT scans did not show any evidence of pneumoconiosis. He explained that the mild pneumoconiosis would not explain the severe defect in pulmonary function studies, the reduction in diffusion, or the drop in PO2 with exercise. Dr. Fino opined the findings were consistent with cigarette smoking induced pulmonary emphysema, possible effects of chemotherapy from colon cancer, and previous lung surgery with resection. Dr. Fino concluded Claimant's pneumoconiosis did not cause or contribute to his disability and that said disability was due entirely to cigarette smoking. He added that there was no evidence of clinical pneumoconiosis.

The supplemental report of Dr. Fino is dated September 23, 2002 and appears at EX 15. He reviewed additional medical records including the medical reports of Drs. Ducatman and Oesterling. He stated that none of the additional information caused him to change any of his opinions previously rendered in this case. He agreed that coal mine dust could cause emphysema but that the drop in FEV-1 was far too large than that predicted by the medical literature. He stated that one would expect a 2-3 cubic centimeter drop in FEV-1 each year due to coal mine dust and a 5 cubic centimeter drop per pack year of cigarette smoking. He opined Claimant lost more FEV-1 to smoking and less due to coal mine dust inhalation. He stated three factors that would make it more likely to have a loss in FEV-1 due to coal mine dust inhalation: (1) prolonged coal dust exposure, (2) abnormality on chest x-ray which is proportional to coal mine dust inhaled, and (3) some evidence of mixed obstructive and restrictive impairment. Dr. Fino opined that even if he accepted all of the premises offered by Dr. Ducatman and if he assumed pneumoconiosis did play some role in Claimant's impairment, he concluded that it was no more than a negligible contribution.

The supplemental report of Dr. Fino is dated October 9, 2002 and appears at EX 16. Dr. Fino was asked by Employer to clarify statements from his previous report. He noted that while he agreed with the literature cited by Dr. Ducatman he did not agree with his conclusions regarding the etiology of Claimant's disability. Dr. Fino opined that coal mine dust inhalation was not a contributing factor in Claimant's impairment or disability. He added that based on a review of the objective evidence, that coal dust inhalation has not played any more than a negligible, or de minimis contribution to Claimant's disabling obstructive vent impairment.

The second supplemental report of Dr. Fino is dated December 2, 2005 and appears at EX 37. He reviewed additional information including the medical reports of Drs. Spagnolo, Gaziano, Cohen and Green and the deposition transcripts of Drs. Alan Ducatman and Barbara Ducatman. He noted that there was no specific type of abnormality that had to be seen on lung function studies to diagnose a coal mine dust related condition. He added that the type of abnormality seen in this case could be related to coal mine dust. He stated that the amount of pneumoconiosis and coal mine dust within the lung tissue could be very predictive of the degree of obstruction and emphysema attributable to coal mine dust. He noted that emphysema had a pathological and clinical meaning. The presence of emphysema in the lungs did not automatically infer respiratory impairment. He went on to discuss several studies relating to emphysema and coal mine dust exposure. He concluded there was no doubt that coal mine dust inhalation caused emphysema. Dr. Fino stated that the key issue was the amount of coal mine dust retained in the lung tissue. He noted that the pathologists offered different assessments on this issue. He stated that the chest x-rays and CT scans suggested a clinically insignificant

deposition of coal mine dust within the lung tissue. However, he noted that the gold standard was pathology. He stated that when pathologists disagree he relied on clinical information and that information supported changes consistent with a smoking-related abnormality. He added that although the duration of coal mine dust exposure did not necessarily predict whether one would have impairment, the 13 year history in this case was on the low end of the likelihood for developing significant respiratory impairment due to coal mine dust. However, Dr. Fino added that if Claimant did have a moderately severe pneumoconiosis as noted by Dr. Green, then he could not exclude coal mine dust as being a contributing factor to Claimant's pulmonary impairment and subsequent disability.

Dr. Prasoon Jain

The progress note by Dr. Jain is dated February 5, 2002 and appears at CX 1. Dr. Jain is Board-Certified in Internal Medicine and Pulmonary Disease. (CX 2). He noted that Claimant was followed in Pulmonary Clinic for chronic shortness of breath and previous history of resection of lung metastasis from colon cancer. Dr. Jain stated that the purpose of the visit was to determine whether Claimant's chronic lung situation may be related to his work in the coal mines. He noted an occupational history of 11 years of coal mine employment and a smoking history of 25 to 30 pack years ending in 1998. Claimant's chief complaints were chronic shortness of breath and cough with sputum production. Physical examination of the chest revealed significant bilateral expiratory rhonchi with few scattered crackles. After reviewing pulmonary function studies and a chest x-ray, Dr. Jain concluded Claimant's emphysema was related to both smoking as well as coal mine dust. There was evidence of bilateral fibrotic changes which were most likely due to CWP. He noted Claimant had severe airflow obstruction and that Claimant's physical activities were significantly restricted due to lung problems. He opined that, in all, Claimant had a combination of emphysema, CWP, and residual of repeated lung infections in the past. There was no evidence of active malignancy in the lungs.

Dr. Samuel Spagnolo

The medical report of Dr. Spagnolo is dated March 19, 2002 and appears at EX 8. Dr. Spagnolo is Board-Certified in Internal Medicine and Pulmonary Disease. (EX 12). He conducted a medical record review at the request of Employer. He noted an occupational history of 11 years of coal mine employment and a 25 to 30 pack year smoking history. Dr. Spagnolo reviewed the medical reports of Drs. Scattaregia, Naeye, Bellotte, and Bush. He concluded that based on the negative chest radiographs and lung tissue findings as described by Drs. Bush and Naeye, Claimant had sub-radiographic simple CWP which was a type of CWP too limited to cause any impairment of lung function. He noted that the emphysema was most consistent with the effects of 20 pack years of cigarette smoking. Dr. Spagnolo opined that the progressive emphysema as well as loss of lung tissue has resulted in his increasing inability to breathe. He added that Claimant's shortness of breath may also be worsened by the presence of cardiac disease as suggested by his paroxysmal nocturnal dyspnea and asthmatic/bronchospastic airway condition that results in wheezing. He concluded Claimant did not have a chronic pulmonary impairment attributable to pneumoconiosis or related to his prior coal mine employment.

The supplemental report of Dr. Spagnolo is dated September 14, 2002 and appears at EX 13. He reviewed additional medical records including the medical reports of Drs. Alan Ducatman and Oesterling. He disagreed with Dr. Ducatman's assertion that individuals without radiographic evidence of pneumoconiosis have sufficient pneumoconiosis to significantly alter lung function noting this statement was misleading and was not supported by medical literature. He stated that none of the additional information would cause him to change any of his previously rendered opinions.

The second supplemental report of Dr. Spagnolo is dated December 3, 2005 and appears at EX 38. Dr. Spagnolo reviewed additional information including the medical reports of Drs. Fino, Gaziano, Green, Cohen and the depositions of Drs. Alan Ducatman and Barbara Ducatman. He concluded that none of the additional information caused him to change any of his previously rendered opinions in this case.

The deposition of Dr. Spagnolo was taken on June 15, 2006 and appears at EX 45. He opined that Claimant's severe obstructive airways disease would prevent him from performing his last coal mine job. He noted that the pulmonary function studies correlate with cigarette smoke induced emphysema. He had never seen bullous emphysema caused by coal dust exposure. He stated that coal mine dust could cause focal emphysema. He reviewed the report of Dr. Cohen. He agreed that there can be obstructive airways disease in coal miners and that this disease was related to the amount of dust in the lungs. He was not aware that coal mine dust caused centrilobular emphysema. He noted that in this case he had no difficulty separating the cigarette smoke induced impairment from that caused by coal mine dust because of the minimal CWP and the presence of bullous emphysema. He opined that the minimal CWP did not cause any reduction in the FEV-1. Dr. Spagnolo opined there was no reason to conclude that any of Claimant's lung disease was attributable to Claimant's work in a steel mill, as a welder, or his stint in the Navy. He agreed that extensive pneumonias could cause scarring of the lung. In reviewing the report of Dr. Cohen he opined that most of Dr. Cohen's reasons for concluding that coal mine dust was contributing to Claimant's disability were non-specific to CWP. He had never seen this degree of impairment associated with pneumoconiosis not visible on x-ray. He opined that Claimant's disability was due to bullous emphysema caused by 30 plus years of cigarette smoking. He concluded that Claimant's CWP did not produce any significant clinical abnormality.

On cross-examination, counsel for Claimant pointed out a series of disparities between the medical records and Dr. Spagnolo's summary of the records. He agreed that the sub-radiographic nature of Claimant's pneumoconiosis was important to his conclusion that pneumoconiosis did not cause any impairment. He stated that it would be rare for Category 1 pneumoconiosis to cause pulmonary abnormalities. He stated that if an x-ray was read as negative or 1/0, as a general statement, he would not attribute impairment to pneumoconiosis. He agreed that Drs. Bush and Naeye described the presence of centrilobular emphysema. He stated that a cardiac condition may have contributed to Claimant's shortness of breath but not to the reduced FEV-1. He stated that coal mine dust caused focal emphysema but not centrilobular emphysema. He stated that Claimant did not have reversible lung disease based on the pulmonary function studies.

Dr. Alan M. Ducatman

The medical report of Dr. Ducatman is dated July 19, 2002 and appears at CX 3. Dr. Ducatman is Board-Certified in Occupational Medicine and Internal Medicine. (CX 4). He examined Claimant on July 8, 2002. He reviewed Claimant's medical history and occupational history and noted a smoking history of 20 pack years ending in 1999. Dr. Ducatman was given additional medical records to review. Physical examination of the lungs revealed marked end-expiratory wheeze most prominently at the right base. Pulmonary function studies and arterial blood gases were also performed. He diagnosed Claimant as having status post metastatic colon cancer, history of emphysema and COPD, CWP with current exacerbation, reflux esophagitis by history, asbestos exposure, and probable silica exposure.

Dr. Ducatman explained that Claimant definitely had pneumoconiosis and that it arose from his coal mine employment. He noted Claimant had a complete impairment by AMA criteria. He further opined that Claimant's pulmonary impairment was significantly related to CWP. He agreed that there were two other risk factors: cigarette smoking and lung cancer with surgical resection. He argued that there was no doubt the CWP was significant in this case. He stated that the simple CWP was the minimum fibrotic condition present because there were only biopsy samples of one small section of the lung. To assert that this represented the maximum disease condition would not be fully justified by data or logic. Even assuming this did represent the full extent of Claimant's medical condition, Dr. Ducatman noted that patients with simple CWP can and usually do have abnormalities and pointed to studies to support his claim. He stated that in the face of clearly disabling respiratory disease he could not understand the clinical or epidemiologic basis for stating that no harm has been caused by CWP. He noted that CWP contributed to COPD and although less important than smoking, it was an important contributing cause of Claimant's respiratory insufficiency with a number of symptoms, findings, and measurements, all present.

The deposition of Dr. Ducatman was taken on November 12, 2002 and appears at EX 25. Prior to the deposition he reviewed additional medical records including the medical reports of Drs. Spagnolo, Barbara Ducatman, Cohen, Green, Fino, and Oesterling. He noted that he preferred Dr. Green's pathology evaluation of the case over Dr. Barbara Ducatman because his was more complete. He stated that black pigment did not have to be present in order to diagnose pneumoconiosis but did require the presence of dust-laden macrophages. He opined that one-third of Claimant's chronic bronchitis was due to coal mine dust and two-thirds due to smoking. Dr. Ducatman stated that the place the biopsy was taken was not advantageous to look for pneumoconiosis. He opined that the slides represented the minimum amount of pneumoconiosis present. He noted that loss in FEV-1 due to coal dust would be similar to that of smoking or about 5 ml per year on average. He did not know whether any of Claimant's impairment was due to asbestos exposure.

Dr. Ducatman testified that a physician with Board-certification in pulmonary medicine was not more qualified than he in determining the cause of Claimant's lung impairment. He explained that occupational physicians were trained to look at etiologic issues and that pulmonologist had far less training in epidemiology, toxicology, and causation. He stated that the presence of reversibility on pulmonary functions did not rule out the presence of

pneumoconiosis, nor did it rule out the presence of an underlying fixed obstruction. He opined that Claimant's reduced pulmonary function studies and arterial blood gases were the result of smoking, pneumoconiosis, and chronic bronchitis. He stated that the negative radiological evidence in this case had no impact because the pathology was the gold standard. He disagreed with the physicians who concluded that Claimant's pneumoconiosis was too mild to have caused impairment. He noted that the epidemiologic data indicated that individuals with CWP have a very substantial exposure to coal dust and that these individuals have very reproducible and well understood losses of pulmonary function over time. Stated differently, coal dust contributes to predictable loss in pulmonary function over time and when exposure is substantial, it results in impairment.

He noted that in the presence of histopathologically- present- pneumoconiosis, individuals have sufficient pneumoconiosis to significantly alter or significantly contribute to lung function abnormalities. Even assuming Claimant had a smoking history of 30-40 pack years, his opinion would not change because he noted a history of 20 pack years or more in his medical report. He did not agree that radiation and chemotherapy contributed to Claimant's fibrosis because Claimant received radiation after the biopsy was taken and he did not believe adequate work was done to show that chemotherapy with this type of cancer caused fibrosis. He did not believe Claimant was a chronic asthmatic but had chronic bronchitis. He opined that 2/3 of Claimant's impairment was due to smoking and 1/3 to coal mine dust.

Dr. Robert Cohen

The medical report of Dr. Cohen is dated October 31, 2002 and appears at CX 10. Dr. Cohen is Board-Certified in Internal Medicine and Pulmonary Disease. (CX 11). He conducted a medical records review at the request of Claimant. He noted an occupational history of 11 years of coal mine employment and a smoking history of 20 to 40 pack years ending in 1999. He summarized the various medical reports in this case. He concluded Claimant had coal workers' pneumoconiosis and that his chronic lung condition was substantially related to his 13 years of coal mine employment, 11 of which were underground, and his 20-40 pack year smoking history. In discussing obstructive lung disease and coal dust, Dr. Cohen noted that medical textbooks included statements that the effects of dust exposure on lung function are significant even after adjusting for smoking habits of miners and that coal mine dust and tobacco smoke produce similar decrements in lung function. He concluded that clearly coal dust causes obstructive lung disease with impairment in FEV-1, like it did to Claimant.

Dr. Cohen took issue with some of the consultants' opinions. He disagreed with Dr. Spagnolo's critique of Dr. Ducatman's report. He noted that Dr. Ducatman had two comments that were very germane to this case: (1) lung biopsy in this case was not representative of the extent of the interstitial lung disease, and (2) documented CWP. He disagreed with Dr. Bellotte's diagnosis of congestive heart failure and asthma noting there was no testing in the record to support either diagnosis. He opined that there was no evidence of pulmonary fibrosis secondary to chemotherapy on biopsy. He noted that the agent used for treatment of the rectal cancer were not those associated with causing pulmonary fibrosis. He disagreed with Dr. Naeye's assertion there was no connection between the development of emphysema and coal mine dust exposure. Dr. Cohen also disagreed with Dr. Fino's statement that thirteen years of

coal mine dust exposure was not long enough to cause disease. He agreed with Dr. Fino's assertion that coal mine dust caused a 2-3 ml decline in FEV-1 per year of underground work as compared to cigarette smoking causing 5 ml loss per year of tobacco smoke. He concluded that coal mine dust was 1/3 to 2/3rds as important as the effect of tobacco smoke. Dr. Cohen added that Claimant was clearly sensitive to pulmonary toxins, coal dust, and tobacco smoke and that it resulted in significant disease.

Finally, Dr .Cohen concluded Claimant's pneumoconiosis clearly rendered him unable to perform the duties of his last coal mine job. He noted that the reduction in FEV-1, severe diffusion impairment, and altered gas exchange was caused by severe obstructive lung disease caused by 13 year exposure to coal mine dust and his 20-40 pack years of exposure to tobacco smoke. He added that the presence of coal macules contributed to this impairment.

The deposition of Dr. Cohen was taken on February 9, 2006 and appears at CX 17. Prior to the deposition, Dr. Cohen reviewed additional medical reports including the reports of Drs. Green, Caffrey, Naeye, Renn, Gaziano, Fino, Spagnolo, Rosenberg, and Alan Ducatman. He assumed a smoking history of 25 to 40 pack years in evaluating the medical evidence. He noted that Claimant's drop in PO2 with exercise was consistent with a coal mine induced disease due to scarring of the alveolar capillary membrane that prevents oxygen from passing through the capillary wall when blood is moving fast through the lungs such as when one is exercising. He added that emphysema was also associated with significant diffusion impairment and could cause hypoxemia with exercise. He noted that the types of CT scans in this case were sub-optimal to rule out pneumoconiosis. Dr. Cohen stated that he based his diagnosis of pneumoconiosis on Claimant's history of exposure, clinical and physical exam findings of chronic lung disease, physiology that was compatible and pathology that was diagnostic of coal workers' pneumoconiosis. He noted that Claimant's lungs showed two types of effects from coal dust: (1) interstitial fibrosis or medical pneumoconiosis and (2) COPD. He stated that radiation and the type of chemotherapy Claimant received would not have caused pulmonary fibrosis. He added that there was no evidence pathologically, radiologically, physiologically or clinically that Claimant had asthma.

Dr. Cohen stated that the literature showed a very significant causal relationship between coal mine dust exposure and the presence of emphysema. These studies have shown the effect of coal mine dust and that lung function impairment is additive to that of cigarette smoking. Dr. Cohen noted that there is good evidence that if one is susceptible to the effects of tobacco you would be also more susceptible to similar toxins such as coal mine dust. It was his opinion that Claimant was more susceptible to both of these exposures. He added that the medical literature demonstrated a causal connection between coal mine dust and centrilobular emphysema. He commented on the study by Dr. Lee noting that this study showed a very significant relationship between coal mine dust content in the lungs and the extent and presence of emphysema. Dr. Cohen noted that bullous emphysema was a continuum of the disease process and was not a different disease than centrilobular emphysema. He opined that Claimant's coal mine dust contributed from 1/3 to 1/2 of Claimant's obstructive impairment. He added that coal mine dust and cigarette smoking were both significantly contributory to Claimant's current pulmonary impairment.

Dr. David M. Rosenberg

The medical report of Dr. Rosenberg is dated December 6, 2005 and appears at EX 39. Dr. Rosenberg is Board-Certified in Internal Medicine, Pulmonary Disease, and Occupational Medicine. (EX 41). Dr. Rosenberg conducted a medical record review at the request of Employer. Dr. Rosenberg reviewed and summarized the medical evidence in the case. He noted that overall, the pathology of the lung sections revealed a minimal degree of simple CWP, associated with minimal degree of focal emphysema. Claimant also had moderate to severe centrilobular emphysema with fibrotic bands. Chest x-rays and CT scans showed an absence of micronodularity with the presence of moderate to severe emphysema. Dr. Rosenberg opined that based on the chest x-rays, CT scans, pulmonary function tests, and arterial blood gases, Claimant would not be diagnosed as having clinical pneumoconiosis. He opined that Claimant had mild pneumoconiosis and that since CWP started in the upper lobes and that was where the samples were taken for the biopsy, his pathologic findings would be considered representative of the lungs generally. He opined that the "fibrosis" described pathologically did not indicate Claimant had diffuse interstitial lung disease related to coal mine dust or any other etiology. He noted that the fibrotic bands were likely due to past inflammatory process or previous lung infection. He noted that Claimant could not perform his last coal mine employment based on his severe airflow obstruction.

Dr. Rosenberg agreed that coal mine dust could cause airflow obstruction but that marked decrease in FEV-1 generally was not consistent with airflow obstruction related to past coal mine dust inhalation but was classic for the presence of smoking-related COPD. He added that the presence of emphysema with bullae formation and low diffusing capacity also supported a smoking-related COPD. He noted that a coal mine dust related impairment would not be associated with a bronchodilator response. Dr. Rosenberg stated that if the severe centrilobular emphysema was related to past coal mine dust inhalation, it would be directly associated with the presence of clinical CWP macules and micronodules. This was not the case here. Dr. Rosenberg concluded Claimant did not have medical or legal pneumoconiosis.[5] He opined Claimant's pulmonary disability was due to smoking-related COPD and that said COPD was not caused or aggravated by coal mine dust.

The deposition of Dr. Rosenberg was taken on January 9, 2006 and appears at EX 44. He agreed that Claimant had sufficient coal mine dust exposure to have caused CWP. He noted a smoking history of 25-30 pack years. He opined that the lung samples were representative of Claimant's lungs generally. He opined Claimant had mild simple CWP based on the clinical data. He noted that focal and centrilobular emphysema was present in the pathology samples. He noted that smoking was the most common cause of centrilobular emphysema. He noted that since the extensive centrilobular emphysema was not associated with macules and micronodules, this would not be emphysema related to coal mine dust. He opined the bullous emphysema was not due to coal mine dust because it did not have the structural changes in and around the bullae that would represent a pathological change due to coal mine dust. He opined that radiation

[5] Although this statement is contradictory to Dr. Rosenberg's prior statement of pathological findings consistent with minimal CWP, I find that Dr. Rosenberg did conclude that Claimant had minimal CWP based on the pathological findings. He repeated his conclusions of minimal CWP based on pathological findings throughout his deposition testimony.

therapy could cause fibrosis in the lungs. He concluded the bronchitis was not due to coal mine dust because Claimant left the mines in 1985, so he would not expect bronchitis in 2000 to be related to coal mine dust. He noted that Claimant's pulmonary function study from 2002 was consistent with asthma. He opined Claimant had a totally disabling pulmonary impairment based on the low FEV-1. He stated that in general if one had mild forms of CWP radiologically or pathologically, pulmonary function studies were preserved with no obstruction or restriction. He opined that CWP was not a substantially contributing cause of Claimant's disability. He stated that one would not expect such a large drop in FEV-1 due to coal mine dust. In contrast, this type of pattern was classic for smoking. He attributed Claimant's total disability to cigarette smoking. He disagreed with Dr. Green's apportionment of 1/3 to 1/2 of the emphysema due to coal mine dust. He noted that you had to look at the entire clinical picture and could not apportion it based on a history of exposure.

Dr. Rosenberg did not review additional medical records prior to the deposition. The last time he reviewed the medical evidence was before December 6, 2005. He opined that the 1993 chemotherapy did not cause any interstitial fibrosis. He stated that his opinion that the biopsy showed only a mild form of CWP was based on a review of all of the pathology reports but that it was not Dr. Green's opinion. He agreed that you could have minimal changes on x-ray and still have disabling COPD. He opined Claimant had minimal pathologic findings of CWP and the emphysema was not related to coal mine dust. He opined that more extensive changes due to CWP would be required to cause impairment. He noted that Claimant received radiation therapy after the biopsy was taken therefore, the radiation did not cause scarring. It was his belief that in general, the FEV-1 of coal miners does not drop to any significant extent. He noted that specific individuals can have significant obstruction but that Claimant did not fall into this category. He based this opinion on Claimant's reduced FEV-1%, markedly reduced diffusing capacity, increased lung volumes and air trapping. In looking at the whole picture, it was classic for cigarette smoking related COPD. He admitted that Claimant experienced some loss of FEV-1 due to coal mine dust (150ccs over 15 years) but classified this as clinically insignificant. He stated that he has had patients with negative chest x-rays with significant obstruction. In those cases he attributed the decline in lung function to coal mine dust because there was no other explanation.

Biopsy/Pathology Reports

Dr. Barbara S. Ducatman

The pathology report of Dr. Ducatman is dated April 6, 1999 and appears at DX 41. A wedge biopsy of Claimant's left upper lobe was performed on April 2, 1999 at West Virginia University Hospital. The final pathologic diagnoses were organizing pneumonia, lymphoid hyperplasia, lipid pneumonia, coal workers' pneumoconiosis, and emphysema. The mass was identified as moderately differentiated adenocarcinoma consistent with colon primary. Pleural fibrosis and five anthracotic lymph nodes were also noted. Dr. Ducatman stated that there was an abundant hemosiderin within the macrophages as demonstrated by iron stains.

The deposition of Dr. Barbara Ducatman was taken on May 11, 2001 and appears at EX 4. This deposition was taken by counsel for Employer. Counsel for Claimant was not present.

Dr. Ducatman is the chair of pathology at West Virginia University. She noted that the lung biopsy was not necessarily a representative sample of the lung because it was taken because of metastasis. She added that pleural fibrosis was a nonspecific finding and that it could be due to emphysema, CWP, pneumonia, and certain chemotherapies. Dr. Ducatman stated she saw a proliferation of coal macules but no nodules. She noted there was some fibrous reaction to the coal dust. Based on the limited information she had, Dr. Ducatman could not say whether Claimant was disabled. She stated that Claimant's emphysema was consistent with cigarette smoking.

The second deposition of Dr. Ducatman was taken on September 25, 2002 and is found at CX 5. Dr. Ducatman is Board-Certified in Anatomic and Clinical Pathology and Cytopathology. Prior to the deposition she reviewed hospital records, medical testing results, the medical reports of Drs. Naeye, Bush, and Oesterling, and the medical reports of Drs. Scattaregia, Bellotte, Fino, and Alan Ducatman. She noted that Alan Ducatman was her husband but that she did not discuss the case with him. Dr. Ducatman stated that based on the pulmonary function tests Claimant's impairment was severe. She concluded that the biopsy slides did not reflect the full extent of Claimant's pneumoconiosis based on the vent studies, diffusing capacity, and exercise blood gases. She opined that pneumoconiosis did contribute to Claimant's pulmonary function decreases. She referenced a study that concluded that there was an association between exposure to respirable coal dust and emphysema in coal miners but only in men who showed some dust-related fibrosis and that such fibrosis might be a factor in determining the presence of centriacinar/centrilobular emphysema. Claimant had such fibrosis. In fact, Dr. Ducatman's own research supported these conclusions. Dr. Ducatman concluded that Claimant's coal mine dust exposure contributed to his centrilobular emphysema. She noted that the absence of black pigment in the areas of emphysema would not mean that coal dust did not play a role in its development because the development of emphysema was more complicated than just laying down of pigment.

On cross-examination Dr. Ducatman stated that until recently she believed that severity of emphysema generally correlated with pack-year history but a new study showed that it did not. She acknowledged that the biopsy was from the upper lobe which was where the most severe portion of the lung was typically affected by CWP. She stated she would defer to a pulmonologist in the interpretation and application of the clinical significance of the pulmonary function testing. She admitted that she could not offer (nor could anyone else) an opinion based on a reasonable degree of certainty based on this pathology as to whether or not Claimant's coal mining had caused a disease that would be causing lifetime impairment. Again this was due to the non-representative sampling of the biopsy wedges.

Dr. Richard Naeye

The medical report of Dr. Naeye is dated January 17, 2001 and appears at DX 41. Dr. Naeye is Board-Certified in Anatomic and Clinical Pathology. He reviewed Claimant's medical records and noted an occupational history of 11 years of coal mine employment. He noted a smoking history of one pack per day for 40 years. He also reviewed 14 slides taken from Claimant's lung biopsy. He stated that the minimal finding to make the diagnosis of very mild simple CWP was present and added that this CWP was far too mild to have caused any

measurable abnormalities in lung function or disability. He noted that Claimant had severe centrilobular emphysema and that the major cause was Claimant's smoking habit. He added that almost all of Claimant's disabling emphysema should be attributed to it rather than coal mine dust. He argued that a critical review of the literature indicated that mine dust has no role or only a clinically insignificant role in the genesis of centrilobular emphysema. Dr. Naeye stated that there were areas of fibrosis that indicated the presence of infection, presumably the consequence of Claimant's repeated bouts with pneumonia. He opined that since these areas did not have any black pigment or tiny birefringent crystals they do not have a CWP origin.

The deposition of Dr. Naeye was taken on April 16, 2001 and appears at EX 2. Counsel for Claimant did not appear. He noted he had adequate tissue and sampling to make a diagnosis of pneumoconiosis. He stated that the biopsied lobe had six rounds of radiation therapy and that chemotherapy also contributed to the tissue damage. Dr. Naeye agreed that Claimant's fibrosis was caused by pneumoconiosis, desmoplastic (reaction to cancer) process, (radiation and chemo) therapy, and bouts of pneumonia. He opined that 98% of the fibrosis was not associated with black pigment. He agreed that coal mine dust can cause centrilobular emphysema but that in a case of severe emphysema less than 5% would be attributable to coal mine dust. In this case the remainder would be caused by smoking, aging, and bouts of pneumonia. He opined that Claimant's CWP was not causing or contributing to his pulmonary impairment because it was too mild to have caused any measurable proportion of the centrilobular emphysema that was present.

The supplemental medical report of Dr. Naeye is dated December 17, 2005 and appears at EX 42. He reviewed additional medical records including the medical reports of Drs. Barbara Ducatman, Alan Ducatman, Fino, Green, Oesterling, Cohen, Spagnolo, Scattaregia, and Gaziano. He did not challenge Dr. Cohen's review of the CWP literature but criticized his failure to recognize, describe, and interpret the specific CWP findings in Claimant's lungs. He noted that Dr. Cohen's report was generic rather than specific. He disagreed with Dr. Green's characterization of the amount of CWP present noting that the fibrosis described by Dr. Green was actually a local phenomenon caused by local infections that resulted from cancer's destruction of lymphatics and other normal defense mechanisms in Claimant's lungs. He added that interstitial fibrosis was not a constituent disorder of CWP unless a miner had very heavy exposures to the very tiny fibrogenic particles of toxic silica. He stated that he stood by his original conclusions that CWP lesions were "too limited in severity and extent to have caused clinically significant abnormalities in lung function and have had a clinically significant role in his death."

Dr. Stephen Bush

The medical report of Dr. Bush is dated February 2, 2001 and appears at DX 41. Dr. Bush is Board-Certified in Anatomic and Clinical Pathology. He reviewed and summarized various medical records and reviewed the lung biopsy slides. He noted a smoking history of 40 pack years. Dr. Bush opined there was sufficient evidence to justify a diagnosis of a minimal degree of simple CWP. He added that Claimant had a respiratory impairment related to COPD resulting from a long history of cigarette smoking. He opined that coal mine dust exposure and the mild degree of CWP was not causally related to any respiratory impairment. He noted that

the degree of CWP was extremely mild and would not have been severe enough to produce symptoms or impairment. This opinion was based on the absence of evidence of pneumoconiosis in multiple chest x-rays and CT scans. Dr. Bush concluded Claimant was totally disabled due to metastatic carcinoma from the rectum to the lung and due to the moderate degree of centrilobular emphysema resulting from smoking. Claimant's disability was not caused in whole or in part by CWP, which was too mild in degree and too limited in extent to be a factor in disability.

Dr. Everett Oesterling

The medical report of Dr. Oesterling is dated September 4, 2002 and appears at EX 10. Dr. Oesterling is Board-Certified in Anatomic and Clinical Pathology. (EX 11). He reviewed the biopsy slides at the request of Employer. Based on his review of the slides he opined there was sufficient evidence to justify a diagnosis of mild macular CWP which was too minimal to have altered pulmonary function. He noted Claimant may have a respiratory impairment but that it could not be attributable to coal mine dust. He added that based on the tissue present, Claimant would not appear to be totally disabled and should be capable of returning to coal mine work. He concluded there was no disability caused by CWP. Dr. Oesterling stated that Claimant's moderate centrilobular emphysema and bronchiolitis was related to cigarette smoking.

The deposition of Dr. Oesterling was taken on June 8, 2006 and appears at EX 46. He testified that Claimant had mild micronodular pneumoconiosis. He described the presence of metastatic cancer, smoker's macrophages, evidence of hemorrhage, and centrilobular emphysema. He agreed with the conclusions in the pathology report of Dr. Barbara Ducatman. He opined that Claimant's emphysema was not caused by coal mine dust because: (1) there was no black pigment, (2) one did not typically see this level of emphysema with this level of CWP, and (3) bullous emphysema was not typically seen in coal workers. He opined that Claimant's emphysema was due to smoking. He opined that Dr. Green was confusing areas of smooth muscle and interstitial fibrosis. He added that a lot of fibrosis was caused by hemosiderin and not coal mine dust. He noted that Dr. Green's photographs showed a much larger area with more deposits than his slides. He opined that the emphysema that he saw on the slides would not have been disabling but that the records reflected the presence of more severe emphysema. It was pointed put that on slide A3, Dr. Oesterling opined in his report that there was nothing on this slide related to coal but that it was all emphysema due to smoking. Dr. Green reviewed the same slide and identified 11 lesions related to coal mine dust. Dr. Oesterling stated that he would attribute 20% of Claimant's centrilobular emphysema due to coal mine dust. He based this percentage on the amount of pigment in the lungs and Claimant's lengthy smoking history. He opined that the centrilobular would have caused a relatively small amount of impairment when compared to that caused by bullous emphysema.

Dr. Francis H.Y. Green

The medical report of Dr. Green is dated October 31, 2002 and appears at CX 8. He is Board-Certified in Anatomic Pathology and his major research interests include occupational pneumoconiosis and the development of diagnostic criteria and standards and the correlation of

pathological, radiological, and physiological aspects of pneumoconiosis. (CX 9). Dr. Green conducted a medical records review and also evaluated the lung biopsy slides at the request of Claimant. Dr. Green's review of the biopsy slides revealed the presence of metastatic adenocarcinoma, simple macular and micronodular CWP of moderate severity, moderate interstitial fibrosis associated with heavy deposits of carbon, silica, silicates and ferruginous bodies consistent with mixed dust pneumoconiosis, severe centriacinar and irregular emphysema, pneumonia, and pulmonary vascular changes. Dr. Green noted an occupational history of 13 ½ years of coal mine employment and a smoking history of 25 to 40 pack years ending in 1999.

Dr. Green noted that he along with Drs. Ducatman, Naeye, Oersterling, and Bush agreed that metastatic cancer was present, that the lungs showed evidence of moderate to severe emphysema, there was evidence of CWP, that the lungs showed varying degrees of interstitial fibrosis and organizing pneumonia, and the presence of pigment in the lungs including black pigment, ferruginous particles, and silica (under polarized light). He noted that the small lung biopsy of the left upper lobe was reasonably representative of the lung as a whole because the extent of the emphysema and CWP was relatively representative of the clinical findings and were consistent with the areas of irregular opacities and scarring noted on chest x-rays.

Dr. Green addressed Dr. Naeye's assertion that there was extensive fibrosis damage done to this lobe by six rounds of radiation therapy and that Claimant's chemotherapy also contributed. Dr. Green acknowledged that radiation could cause fibrosis of the lung but noted that Claimant did not have radiation or chemotherapy until *after* the biopsy was taken. Claimant did have radiation and chemotherapy in 1993 and 1994 but clearly by his wording, Dr. Naeye was not referring to these treatments. Dr. Green noted that in 1993/1994 there was no evidence of metastatic disease in the lungs therefore radiation would not have been directed to the lung but to the lymph nodes in the abdomen and pelvis. He concluded therefore that radiotherapy given in 1993/1994 could not have been a factor in producing lung fibrosis.

Dr. Green reviewed Dr. Oesterling's photomicrographs and stated that these photos illustrate the cancer and emphysema adequately but fail to show pneumoconiosis (macules, micronodules, or interstitial fibrosis).

Dr. Green reviewed the radiographic evidence and noted that x-ray changes in pneumoconiosis were non-specific and most radiologists gave a descriptive report only. He noted that the radiograph is less sensitive than pathology in detecting pneumoconiosis. Moreover, in the presence of emphysema, the x-ray appearances of pneumoconiosis are attenuated leading to under-reading of the category of pneumoconiosis. He concluded that the radiologic observations in this case were not in conflict with the pathologic findings.

He discussed COPD and Claimant's exposure to coal mine dust. He noted that autopsy studies looking at the relative contribution of cigarette smoking and coal mine dust exposure to the development of emphysema indicate that one pack year of cigarette smoking causes approximately the same amount of emphysema as one year of underground coal mining. Dr. Green concluded that the evidence overwhelmingly implicates coal mine dust exposure in the development of emphysema. Taking into account Claimant's smoking history of 25-40 pack years, he estimated that coal mine dust caused approximately 1/3 to 1/2 of Claimant's

emphysema and chronic bronchitis. Dr. Green concluded that medical pneumoconiosis resulted in whole from exposure to coal mine dust in the coal mines and contributed to the impairment in diffusing capacity and gas exchange. Furthermore he stated that coal mine dust contributed about 1/3 to 1/2 to Claimant's obstructive lung disease (chronic bronchitis and emphysema). It was his view that dust exposure was a major cause of Claimant's respiratory impairment.

The deposition of Dr. Green was taken on January 17, 2006 and appears at CX 16. Dr. Green described his background and the ongoing research he is involved in related to occupational lung diseases of coal miners. He noted that the research showed that one year of underground mining produced, approximately the same amount of emphysema as a year of smoking on average. He added that some individuals are more susceptible than others to the effects of inhaling coal mine dust and cigarette smoke. Dr. Green noted that if you were susceptible to smoke then you would be susceptible to coal mine dust since it was the same mechanism at play regardless of the irritant. He stated that centrilobular, centriacinar, and focal emphysema were all the same thing. He noted that one person inhaling dust may get predominately pneumoconiosis, another person may get predominately emphysema, and a lot of miners would get both. Dr. Green stated that he was unaware of any study that refuted the association between coal mine dust and the development of chronic obstructive lung disease including emphysema. He noted that the studies on smoking and emphysema were conducted in the 1960s and 1970s and that none of them had a control for occupational exposure.

Dr. Green reviewed additional medical records before the deposition including the reports of Drs. Fino, Rosenberg, Spagnolo, Caffrey, Naeye, and Cohen. None of the records caused him to change any of his opinions. He reviewed the photomicrographs attached to his medical report. He noted that based on Dr. Oesterling's photomicrographs he would not be able to diagnose pneumoconiosis at all. Dr. Green opined that Claimant had pneumoconiosis partly due to the fibrotic classic lesions of medical pneumoconiosis and partly due to the dust-induced emphysema and that together these were enough to seriously contribute to his impairment. He noted that Claimant did not have radiation to his lungs prior to the biopsy and did not have the type of fibrosis that would be seen due to chemotherapy. He disagreed with Dr. Naeye's opinion that there had to be a large number of silica crystals in order for interstitial fibrosis to be attributable to coal mine dust. He disagreed with Dr. Rosenberg's opinion that centrilobular emphysema could not be attributable to coal mine dust unless there were macules and micronodules of pneumoconiosis associated with the emphysema. He noted that a study by Dr. Lee showed that coal mine dust, not pneumoconiosis, was related to emphysema explaining why in a given miner the amounts of one or the other will be different. Dr. Green stated that he conducted a short study that showed that coal mine dust exposure was associated with all types of emphysema but particularly focal and centriacinar.

Based on his review of the biopsy slides, the most significant finding was the presence of metastatic cancer, followed by emphysema, and pneumoconiosis. He found no evidence of asthma or granulomatous disease. Dr. Green stated that his own studies showed a strong relationship between panacinar emphysema and coal mining but not as strong as with centriacinar. He added that Claimant's cigarette smoking was responsible for only a minimal amount of interstitial fibrosis present in the miner. Dr. Green noted that his new data confirmed the relationship between bullous emphysema and coal mine dust.

Dr. P. Raphael Caffrey

The medical report of Dr. Caffrey is dated December 9, 2005 and appears at EX 40. Dr. Caffrey is Board-Certified in Anatomical and Clinical Pathology. (EX 41). He conducted a medical record review at the request of Employer. He also examined the tissue slides from the lung biopsy. He noted the presence of simple pneumoconiosis, mild interstitial fibrosis, moderate emphysema, adenocarcinoma, and focal subpleural fibrosis. He noted a smoking history of 24 to 40 pack years ending in 1999. Dr. Caffrey diagnosed Claimant as having simple CWP but opined Claimant did not have legal pneumoconiosis based on evidence in the record that Claimant suffered from asthma and chronic bronchitis. He opined these conditions were not caused by coal mine dust because Claimant continued to have bronchitis long after he was out of the mines. He opined Claimant's emphysema was due mainly to his 25-40 pack year history of smoking. He stated that all clinicians agree that smoking is the number one cause of emphysema. He agreed that coal mine dust could cause emphysema in a susceptible individual who has been exposed to coal mine dust over a proper length of time. He noted that Claimant's 11 years of underground coal mine employment was a minimal amount of time for someone to develop CWP and emphysema. He acknowledged that the slides showed the presence of simple CWP so he concluded that some of the emphysema could be due to coal mine dust. He stated that, based on the minimal findings on chest x-ray and CT scans, one would not expect to have a severe disability due to mild or minimal CWP. He stated that he agreed with Dr. Barbara Ducatman's opinion that only 5% of Claimant's emphysema was due to coal mine dust.

Dr. Caffrey reviewed the opinion of Dr. Alan Ducatman. He referenced a comment by Dr. Ducatman in his medical report that stated, "[Claimant's] pulmonary impairment is significantly related to coal workers' pneumoconiosis." Dr. Caffrey noted that Dr. Ducatman did not provide any objective data to justify a diagnosis of "significantly." He went on to state that the word "significant" to him meant approximately 50% or greater cause of the particular disease in question, which in this case was emphysema. He opined that certainly CWP was not the major cause of emphysema in this case. He noted that a needle biopsy in 2001 showed cellular changes consistent with granulomatous process and that there was no cause and effect between granulomatous disease and coal mine employment.

He noted that Claimant's chemotherapy and radiation in 1993 would not have had an effect on the wedge of lung that was removed in 1999. He noted however that subsequent radiation and chemotherapy could be having an effect on the lung tissue. He disagreed with Dr. Green's assessment that 1/3 to 1/2 of Claimant's emphysema/bronchitis was due to coal mine dust. He noted the paucity of readings for CWP and Dr. Barbara Ducatman's assessment of 5% of emphysema due to CWP. He concluded that 5% would not be sufficient to cause a significant or discernable disability. He opined that claimant's pulmonary disability was due to Claimant's 25-40 pack year smoking plus the carcinoma of the colon which had metastasized to the lung causing Claimant to undergo two rounds of radiation and chemotherapy in 1993 and 1999. In addition, Claimant had suffered frequent bouts of pneumonia. Dr. Caffey opined that if Claimant had not been a smoker and did not have carcinoma of the colon, Claimant would not have any discernable disability.

Miscellaneous Medical Records

The records contains and I reviewed the medical records from West Virginia Hospital (DX 38; EX 26), Veteran's Area Medical Center (DX 37; EX 3; 27; 36); Dr. Richard Piccirillo (DX 44); and Davis Memorial Hospital (DX 38).

Conclusions of Law

Length of Coal Mine Employment

The parties stipulated and I find that Claimant was a coal miner, within the meaning of the Act, for 11 years. (TR 10).

Date of Filing

I find that Claimant filed his claim for benefits under the Act on June 8, 2000. (DX 1).

Responsible Operator

I find the evidence of record supports the conclusion that Elkay Mining Company is the properly named responsible operator in this case. (TR 10-11).

Dependents

I find that Claimant has one dependent for purposes of augmentation of benefits under the Act, his wife. (TR 10-12).

Standard of Review

The administrative law judge need not accept the opinion of any particular medical witness or expert, but must weigh all the evidence and draw his/her own conclusions and inferences. *Lafferty v. Cannelton Industries, Inc.*, 12 B.L.R. 1-190 (1989); *Stark v. Director, OWCP*, 9 B.L.R. 1-36 (1986). The adjudicator's function is to resolve the conflicts in the medical evidence; those findings will not be disturbed on appeal if supported by substantial evidence. *Lafferty, supra; Short v. Westmoreland Coal Co.*, 10 B.L.R. 1-127 (1987); *Piccin v. Director, OWCP*, 6 B.L.R. 1-616 (1983).

In considering the medical evidence of record, an administrative law judge must not selectively analyze the evidence. See *Wright v. Director, OWCP*, 7 B.L.R. 1-475 (1984); *Hess v. Clinchfield Coal Co.*, 7 B.L.R. 1-295 (1984); *Crider v. Dean Jones Coal Co.*, 6 B.L.R. 1-606 (1983); see also *Stevenson v. Windsor Power House Coal Co.*, 6 B.L.R. 1-1315 (1984). The weight of the evidence, and determinations concerning credibility of medical experts and witnesses, however, is for the administrative law judge. *Mabe v. Bishop Coal Co.*, 9 B.L.R. 1-67 (1986); *Brown v. Director, OWCP*, 7 B.L.R. 1-730 (1985); see also *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985); *Henning v. Peabody Coal Co.*, 7 B.L.R. 1-753 (1985).

As the trier-of-fact, the administrative law judge has broad discretion to assess the evidence of record and determine whether a party has met its burden of proof. *Kuchwara v. Director, OWCP*, 7 B.L.R. 1-167 (1984). In considering the evidence on any particular issue, the administrative law judge must be cognizant of which party bears the burden of proof. Claimant has the general burden of establishing entitlement and the initial burden of going forward with the evidence. See *White v. Director, OWCP*, 6 B.L.R. 1-368 (1983).

Entitlement: In General

To establish entitlement to benefits, a claimant must establish that he has pneumoconiosis, that his pneumoconiosis arose out of coal mine employment, that he is totally disabled, and that his total disability is due to pneumoconiosis.

Employer conceded in its post-hearing brief that Claimant had pneumoconiosis arising out of his coal mine employment and that Claimant had a totally disabling pulmonary impairment. After a thorough review of the medical evidence, I find that the overwhelming evidence affirmatively establishes the presence of coal workers' pneumoconiosis due to coal mine dust exposure and the presence of a totally disabling pulmonary impairment. Therefore, the sole issue to be decided is whether Claimant's pneumoconiosis was a substantially contributing factor to his totally disabling pulmonary impairment.

Disability Causation

The final issue is whether Claimant has established disability causation at Section 718.204.

Pursuant to § 718.204(c)(1) a miner shall be considered totally disabled due to pneumoconiosis if pneumoconiosis . . . is a substantially contributing cause of the miner's totally disabling respiratory impairment. Pneumoconiosis is a "substantially contributing cause" of the miner's disability if it:

(i) Has a material adverse effect on the miner's respiratory or pulmonary condition;
or
(ii) Materially worsens a totally disabling respiratory or pulmonary impairment which is caused by a disease or exposure unrelated to coal mine employment.

There are fifteen physicians who have rendered an opinion relative to this issue in this case. In general, all of the physicians have diagnosed the presence of pneumoconiosis and that Claimant suffered from a totally disabling pulmonary impairment. The disagreement is over how much pneumoconiosis is present and to what extent the pneumoconiosis is contributing to Claimant's acknowledged total pulmonary disability.

It is well-recognized that pathology is the gold standard in diagnosing the presence of pneumoconiosis and in determining the extent of the disease. Fortunately, in this case there was

a lung biopsy. Of the fifteen physicians who rendered an opinion, six (Drs. Green, Barbara Ducatman, Naeye, Bush, Oesterling, and Caffrey) are pathologists who have examined tissue taken from the lung biopsy.

I accord great weight to the highly qualified opinion of Dr. Green. I find that his opinion is well-reasoned, well-documented, and is consistent with the objective medical evidence, Claimant's occupational history, smoking history, and medical history. He found that the biopsy sample was reasonably representative of the lungs. Based on his extensive review of the biopsy slides he diagnosed Claimant as having simple macular and micronodular CWP of moderate severity. At his deposition, he persuasively explained and defended his diagnoses. He opined that Claimant's pneumoconiosis was due in part to classic fibrotic lesions (clinical pneumoconiosis) and due in part to dust-induced emphysema (legal pneumoconiosis) and that together this was enough to seriously contribute to Claimant's pulmonary impairment. He noted that his research showed that one year of underground coal mining produced about the same amount of emphysema as one pack year of smoking.[6] He reasoned that if one were susceptible to smoke then one would also be sensitive to coal mine dust since it was the same mechanism at play regardless of the irritant. He opined that based on the foregoing that about 1/3 to 1/2 of Claimant's emphysema and chronic bronchitis was due to coal dust exposure. He concluded that coal mine dust was a major cause of Claimant's respiratory impairment. I find this reasoning extremely persuasive.

I accord less weight to the highly-qualified opinion of Dr. Barabara Ducatman. At her second deposition, after some equivocal and inconsistent statements, Dr. Ducatman ultimately concluded that due to the non-representative sampling of the biopsy wedges, she could not render an opinion as to whether or not Claimant's coal mining had caused a disease that would be causing lifetime impairment. Because Dr. Ducatman could not render an opinion with a reasonable degree of medical certainty on the ultimate issue in this case, I accord less weight to her opinion.[7]

That being said, I find that Dr. Ducatman did make some convincing statements regarding the connection between coal mine dust and the formation of centrilobular emphysema. She noted that research confirmed an association between coal mine dust and the formation of

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[6] Claimant's smoking history varied in the records from 20 pack years up to 40 pack years. He testified at his hearing that he smoked less than one pack of cigarettes per day from 1955 to 1999 (44 years) but that he stopped smoking at least five times during that period and that each time he quit it was for several years. (TR 27-28). Claimant opined that he smoked about 25 years. (TR 27-28). Claimant explained that the high end of the range (40 pack years) came from Dr. Graber and that he did not mention to Dr. Graber that he stopped smoking intermittently over the years. (TR 27-28). I find Claimant's testimony credible. Assuming that Claimant quit smoking five times for several years over 44 years and that "several" meant two to three years, I find that Claimant had a history of smoking less than a pack of cigarettes per day for 29 to 34 years.

[7] During her second deposition, Dr. Ducatman stated in response to a question by counsel for Claimant she could not precisely quantify the amount of impairment caused by CWP but agreed that it was more than de minimis. She later said in response to a question from counsel for Employer that de minimis to her meant 5% but that she still was not comfortable with any number. (CX 5). In later depositions and a few medical reports, this exchange was characterized as an endorsement by Dr. Ducatman that only 5% of Claimant's emphysema was due to coal mine dust. I have carefully re-read the deposition transcript and find that Dr. Ducatman never stated that 5% of Claimant's emphysema was due to coal mine dust.

emphysema in coal miners who had evidence of dust-related fibrosis. Since Claimant had evidence of this type of fibrosis she concluded that Claimant's coal mine dust exposure contributed to the formation of centrilobular emphysema (legal pneumoconiosis). Employer may argue that Dr. Ducatman's opinion is equivocal since in her first deposition she opined that Claimant's emphysema was consistent with cigarette smoking. However, I find that prior to her second deposition, Dr. Ducatman reviewed additional medical records that enabled her to have a more complete picture of Claimant's medical condition and that her subsequent testimony reasonably evolved based on the new information she was given access to. I find that Dr. Ducatman's recent research in this area lends further support to the well-reasoned opinion of Dr. Green that at least some of Claimant's emphysema was caused by coal mine dust.

I accord less weight to the highly-qualified opinion of Dr. Naeye on this issue. He diagnosed the presence of mild pneumoconiosis and severe centrilobular emphysema. He argued in his report that a critical review of the literature indicated that mine dust had no role or only a clinically insignificant role in the genesis of centrilobular emphysema. At his deposition, Dr. Naeye acknowledged coal mine dust can cause centrilobular emphysema but stated, as a general rule, that in the case of severe emphysema less than 5% would be due to coal mine dust. He added that long term exposure to coal mine dust can be a contributing factor to the development of centrilobular emphysema but that the contribution <u>was always less than 5%</u>. (EX 2, page 18)(*emphasis added)*. This view is contrary to comments to the Regulations that point to studies that show that centrilobular emphysema was *significantly* more common among coal workers and that the severity of the emphysema was related to the amount of dust in the lungs. Moreover, the Department concluded that medically significant emphysema was related to coal mine dust exposure even in the absence of progressive massive fibrosis. 65 *Federal Register 245, December 20, 2000, 79941-42*.

Dr. Naeye also opined that Claimant's fibrosis was due to pneumoconiosis, reaction to cancer, radiation and chemotherapy, and bouts of pneumonia. However, as Dr. Green discussed in his medical report, Claimant did not receive radiation to the lungs until *after* the biopsy was taken. Therefore, none of the fibrosis viewed by Dr. Naeye on the biopsy slides could have been due to radiation therapy. Moreover, at the end of his supplemental report he noted that the CWP was too limited in severity and extent to have caused significant abnormalities in lung function and have a "clinically significant role in his death." I am sure Claimant, who is very much alive, would find this statement curious. These oversights by Dr. Naeye make me question the thoroughness of his medical record review and the care he took in preparing his report. For the foregoing reasons, I accord his opinion less weight.

I accord less weight to the highly-qualified opinion of Dr. Bush. He diagnosed Claimant as having mild simple CWP, metastatic carcinoma, and moderate centrilobular emphysema due to smoking. He opined that the degree of CWP was extremely mild and would not have been severe enough to have produced symptoms or impairment. He based this opinion on the absence of pneumoconiosis in chest x-rays and CT scans. I find that Dr. Bush's opinion is not well-reasoned and is not well-documented. Although he diagnosed the presence of clinical pneumoconiosis he did not explain how he was able to eliminate coal mine dust as a cause of Claimant's moderate emphysema (legal pneumoconiosis). Moreover, Dr. Bush seems to indicate that he needed to see radiological evidence of pneumoconiosis before he would attribute any

impairment to said disease. However, as Dr. Green explained in his report, in the presence of emphysema the x-ray appearances of pneumoconiosis are attenuated leading to an under-reading of the category of pneumoconiosis. Therefore, it is likely Claimant had a more significant degree of pneumoconiosis than what was being reported on chest x-ray. This is supported by the findings of Dr. Green who found a moderate degree of CWP based on the biopsy slides. Based on the foregoing, I find that eliminating CWP as a source of impairment based on negative chest x-rays and/or CT scans is not credible especially when there is pathologic evidence of the disease.

I accord less weight to the highly-qualified opinion of Dr. Oesterling. I find that his opinion regarding the presence of legal pneumoconiosis was not well-reasoned. In his medical report, he diagnosed Claimant as having simple CWP that was too mild to cause impairment and moderate centrilobular emphysema that he attributed to smoking. At his deposition he opined that Claimant's emphysema was not caused by coal mine dust because (1) there was no black pigment, (2) it was not typical to see this level of emphysema with this mild level of CWP, and (3) bullous emphysema was not caused by coal mine dust.

I will address each of these points. As for the absence of black pigment, Dr. Barbara Ducatman persuasively testified that the absence of black pigment in the areas of emphysema would not mean that coal dust did not play a role in its development because the development of emphysema was more complicated than just the laying down of pigment. As for the ratio of emphysema to CWP, the Act does not require coal mine dust to be the sole source of the impairment. Coal mine dust need only be a contributing factor to the impairment. Based on Claimant's history of exposure to tobacco smoke and studies that clearly link smoking to the formation of emphysema, one can reasonably conclude that at least some of the emphysema would be due to smoking. Therefore, because there are other risk factors at play in this case for the development of emphysema, I find that it would not be appropriate to eliminate coal mine dust as a cause of the emphysema just because the degree of emphysema was more severe than what one would expect in the case of mild CWP. As for the presence of bullous emphysema, Dr. Green testified that new data from a recent study confirmed the relationship between bullous emphysema and coal mine dust. Therefore, I find that the presence of bullous emphysema would not be an appropriate basis to eliminate coal mine dust as a cause of the emphysema.

I also find that Dr. Oesterling's opinion is equivocal. After stating in his medical report and deposition that Claimant's emphysema was due to smoking, he changed his mind and agreed on cross-examination that coal mine dust could cause emphysema and that approximately 20% of Claimant's emphysema was due to coal mine dust.

Moreover, there is persuasive evidence that Dr. Oesterling under-represented the amount of CWP that was actually present. Dr. Green reviewed the photomicrographs taken by Dr. Oesterling and stated that these photographs illustrated the presence of cancer and emphysema but failed to show pneumoconiosis. At his deposition, Dr. Oesterling acknowledged that on one slide he noted that there was nothing related to coal mine dust but that it was all emphysema due to smoking. Dr. Green reviewed the same slide and found 11 lesions related to coal mine dust. Based on the foregoing, I find the opinion of Dr. Oesterling is not credible and is entitled to less weight.

I accord less weight to the opinion of Dr. Caffrey. I find that his opinion is not well-reasoned and is not well-documented. He diagnosed Claimant as having mild simple CWP, mild interstitial fibrosis, and moderate emphysema. He acknowledged that since CWP was present, some (5%) of the emphysema could be due to coal mine dust (legal pneumoconiosis) but did not provide any convincing rationale as to how he arrived at that percentage.[8] He added that 5% would not cause significant or discernable disability. He noted that based on minimal findings on chest x-ray and CT scan one would not expect to have severe disability due to mild CWP. This may arguably be true but the Act does not require CWP to be the sole cause of disability. It need only be a substantial contributing factor. Therefore, even though one might not expect mild CWP to be the sole source of disability in this case, it certainly does not rule it out as a substantial contributing factor. For these reasons, I accord his opinion less weight.

Pulmonology/Internal Medicine Reports

There are nine pulmonary physicians who have rendered an opinion in this case.

I accord great weight to the highly qualified opinion of Dr. Cohen in this matter. His opinion is well-reasoned and is well-documented and is consistent with the objective evidence of record, Claimant's smoking history, occupational history, medical history, and the pathology findings of Dr. Green. He opined that Claimant's reduction in FEV-1, severe diffusion impairment, and altered gas exchange was caused by severe obstructive lung disease caused by 13 years of coal mine dust and his 20-40 pack year exposure to smoking. He stated that studies showed that coal mine dust and tobacco produced similar decrements in lung function. Like Dr. Barabara Ducatman, Dr. Cohen stated that the medical literature showed a causal connection between coal mine dust and centrilobular emphysema. He convincingly explained that the effects of coal mine dust on lung function were additive to that caused by smoking. Moreover, he noted that there was good evidence that if one was susceptible to the effects of tobacco you would also be more susceptible to similar toxins such as coal mine dust. It was his opinion that Claimant was more susceptible to these exposures. Dr. Green, in his report, rendered a similar opinion. He also opined that 1/3 to 1/2 of Claimant's impairment was due to coal mine dust. He added that both coal mine dust and cigarette smoking were both significantly contributory to Claimant's pulmonary impairment. I find the foregoing argument to be reasonable, credible, and highly persuasive. Moreover, Dr. Cohen's opinion and conclusions are supported by the well-reasoned opinion of Dr. Green.[9]

I accord great weight to the highly-qualified opinion of Dr. Alan Ducatman. In addition to being Board-Certified in Internal Medicine, Dr. Ducatman is Board-Certified in Occupational Medicine. He explained at his deposition that occupational physicians such as himself are trained to look at etiologic issues and that pulmonologists had far less training in epidemiology, toxicology, and causation. *Burns v. Director, OWCP*, 7 B.L.R. 1-597 (1984). Moreover, I find

[8] Dr. Caffrey stated that he agreed with Dr. Barbara Ducatman's opinion that 5% of Claimant's emphysema was due to coal mine dust. As I discussed earlier, Dr. Ducatman never made such an assertion.

[9] Dr. Naeye, in his supplemental report, criticized Dr. Cohen's opinion as being generic rather than specific. I disagree. I find that Dr. Cohen, in his report and deposition, pointed to sufficient facts in support of his opinions.

that Dr. Ducatman's opinion is well-reasoned and well-documented and consistent with the reports of Drs. Cohen and Green. He opined that 1/3 of Claimant's pulmonary impairment was due to coal mine dust and 2/3 to smoking.[10] He disagreed with physicians who concluded Claimant's CWP was too mild to cause impairment and explained that data indicated that individuals with CWP have a very substantial exposure to coal mine dust and that these individuals have very reproducible and well-understood losses of pulmonary function over time. He added that in the presence of histopathologically- present- pneumoconiosis individuals, such as Claimant, have sufficient pneumoconiosis to significantly alter or contribute to lung function abnormalities. I find the foregoing opinions to be persuasive and supportive of the opinions of Drs. Green and Cohen.

In addition, in weighing the medical evidence of record, the adjudication officer must give consideration to the relationship between the miner and any treating physician whose report is admitted into evidence. Factors to consider include the nature of the relationship, duration of the relationship, frequency of treatment, and extent of treatment. § 718.104(d). In appropriate cases, the relationship between the miner and his treating physician may constitute substantial evidence in support of the adjudication officer's decision to give that physician's opinion controlling weight, provided that the weight given to the opinion of a miner's treating physician shall also be based on the credibility of the physician's opinion in light of its reasoning and documentation, other relevant evidence and the record as a whole. § 718.104(d)(5). Dr. Jain was one of Claimant's treating physicians and provided a medical report in this matter.

At the hearing Claimant testified that he had been treating with Dr. Jain every three to four months since 2000 for his breathing problems. (TR 25). He added that Dr. Jain prescribed breathing medications for him. (TR 26). Dr. Jain is Board-Certified in Internal Medicine and Pulmonary Disease. (CX 2). Employer has offered no evidence to the contrary. Based on the foregoing, I find that Dr. Jain is a highly qualified physician and pulmonologist who has been treating Claimant for his breathing problems every three to four months over at least a six year period beginning in 2000. Moreover, I find that Dr. Jain prescribed breathing medications for Claimant. Accordingly, I find that the opinion of Dr. Jain would be entitled to special consideration under the "treating physician" rule if his opinion is found to be credible.

I find, however, that the opinion of Dr. Jain is not well-reasoned and is not well-documented. After an examination of Claimant and a review of pulmonary function studies and chest x-rays, Dr. Jain concluded Claimant had emphysema that was related to smoking and coal mine dust. He did not offer an explanation or rationale as to how he arrived at that conclusion. He opined that Claimant's physical activities were significantly restricted due to lung problems and that Claimant had a combination of emphysema, CWP, and residual of repeated lung infections in the past. He did not specifically state that Claimant had a totally disabling lung disease but, assuming arguendo that he did, I find that he did not state to what degree CWP and emphysema caused by coal mine dust (legal pneumoconiosis) contributed to said disability. Because of the foregoing, I accord less weight to the opinion of Dr. Jain on this issue.

[10] Dr. Caffrey was critical of Dr. Alan Ducatman's conclusion in his medical report that stated, "[Claimant's] pulmonary impairment is significantly related to coal workers' pneumoconiosis." Dr. Caffrey argued that Dr. Ducatman provided no objective data to quantify "significantly." I disagree. As noted above, Dr. Ducatman clearly opined that 1/3 of Claimant's impairment was due to coal mine dust and to him this contribution was "significant."

I accord less weight to the highly-qualified opinion of Dr. Gaziano on this issue. First, with an occupational history of 8 ½ years of coal mine employment, he opined Claimant did not have sufficient occupational exposure to explain total disability. Then when he was given a revised occupational history of 13 ½ years of coal mine employment he concluded without explanation that Claimant's impairment was due to a significant degree to occupational exposure to coal mine dust. He later conducted a medical record review and concluded that Claimant's coal mine employment of sufficient duration, the presence of clinical CWP, and pulmonary function study results verifying severe impairment equaled disability caused or contributed to by underground coal mining. Dr. Gaziano seems to be stating that he would presume that a miner's total pulmonary impairment was caused by coal mine dust in any case where there was sufficient occupational exposure, the confirmed presence of CWP, and severe impairment verified by vent studies. I find this presumption is not the standard for causation set forth by the Act. Based on the foregoing, I accord the opinion of Dr. Gaziano less weight.

I accord less weight to the opinion of Dr. Rosenberg. Like Dr. Alan Ducatman, Dr. Rosenberg is Board-Certified in Occupational Medicine. However, I find that his opinion is not well-reasoned and is not-well documented. He opined that Claimant's pulmonary disability was due to smoking-related COPD and that said COPD was not caused or aggravated by coal mine dust. He diagnosed the presence of focal and centrilobular emphysema but concluded that since the extensive centrilobular emphysema was not associated with macules and micronodules, this emphysema was not related to coal mine dust. I find this argument unconvincing. As Dr. Barabara Ducatman persuasively pointed out at her second deposition, the absence of black pigment in the areas of emphysema would not mean that coal mine dust did not play a role in its development because the development of emphysema was more complicated than the laying down of pigment. Even if one were to accept Dr. Rosenberg's assertion, Dr. Green, in fact, had identified in his photomicrographs no. 1 and 4 the presence of centrilobular emphysema adjacent to coal macules and nodules.

Dr. Rosenberg adopted the view that there was minimal CWP present pathologically. He agreed that Dr. Green had a different interpretation but did not say why he chose to reject the findings of Dr. Green in favor of the other pathologists. Dr. Rosenberg opined that, in general, the FEV-1 of coal miners did not drop to any significant extent.[11] He opined that Claimant experienced a loss of 150 ccs over 15 years due to coal mine dust which he classified as clinically insignificant. However, according to Drs. Fino, Cohen, and Alan Ducatman, one would expect, in general, a 3 to 5 cc drop in FEV-1 per year due to smoking. Therefore, in this case, adding the expected drop in FEV-1 due to coal mine dust and cigarette smoking together does not account for the severe obstructive impairment. Dr. Rosenberg assumes that the remainder had to be from smoking. I do not find this conclusion persuasive.

I find the reasoning of Drs. Cohen and Alan Ducatman to be more compelling. They explain that the drop in FEV-1 per year, in general, is about the same for each risk factor. If the

[11] This statement is contrary to the view adopted by the Department of Labor. In the comments to the Regulations it states, "Even in the absence of smoking, coal mine dust exposure is clearly associated with clinically significant airways obstruction and chronic bronchitis. The risk is additive with cigarette smoking." 65 *Federal Register 245, December 20, 2000, 79940*.

total drop in FEV-1 is dramatically greater than expected then the difference has to do with the susceptibility of the individual. If an individual is more susceptible to the effects of cigarette smoke then it follows that they should be more susceptible to the effects of coal mine dust since each follows the same mechanism. Based on this logic it is hard to understand how Dr. Rosenberg is able to minimize the contribution of coal mine dust to the decline in FEV-1. Based on the foregoing, I accord the opinion of Dr. Rosenberg less weight.

I accord less weight to the highly-qualified opinion of Dr. Spagnolo. I find that his opinion is not-well reasoned and is not well-documented. He opined that Claimant's impairment was due, in part, to progressive emphysema as well as loss of lung tissue. He opined that Claimant's sub-radiographic CWP was too limited to cause any impairment in lung function. Contrary to the comments to the Regulations[12] and the opinions of Drs. Fino, Cohen, Rosenberg, Barbara Ducatman, Naeye, Oesterling, Green, and Caffrey, Dr. Spagnolo stated that he was not aware that coal mine dust caused centrilobular emphysema. Since emphysema is an important contributor to Claimant's overall pulmonary impairment in this case, it is important to know the cause of the emphysema. I find that the overwhelming evidence of record supports the assertion that at least some of Claimant's centrilobular emphysema is due to coal mine dust. The fact that Dr. Spagnolo is unable or unwilling to attribute any of the formation of the emphysema to coal mine dust seriously diminishes his credibility on this issue of etiology.

Equally disturbing is the fact that Dr. Spagnolo concluded Claimant's minimal CWP did not cause *any* reduction in the FEV-1. He also stated that it would be *rare* for Category 1 CWP to cause pulmonary abnormalities. Again, these opinions are contrary to the view adopted by the Department of Labor and most of the physicians who rendered an opinion in this case. For these reasons, I accord the opinion of Dr. Spagnolo less weight.

I accord less weight to the opinion of Dr. Scatteregia. Unlike the other consultants in this case, Dr. Scattergia's credentials are less impressive. He is Board-Eligible in Internal Medicine with no certification in pulmonary medicine. In his original report he questioned the presence of CWP and concluded Claimant's pulmonary impairment was due to chronic bronchitis due to smoking and pulmonary fibrosis. After reviewing the pathology report of Dr. Naeye at the request of Employer, he opined Claimant had very slight simple CWP. At his deposition, Dr. Scatteregia basically adopted the opinion and conclusions of Dr. Naeye and without seeing the report of Dr. Bellotte stated he would defer to his expertise. As discussed above, I accorded the opinion of Dr. Naeye less weight especially when compared to the well-reasoned report of Dr. Green. Because Dr. Scatteregia did not have access to other pathology reports in the record and only had limited medical evidence to review, I find that the opinion of Dr. Scatteregia should be accorded less weight.

I accord less weight to the highly-qualified opinion of Dr. Bellotte. In his medical report he concluded there was insufficient evidence to justify a diagnosis of CWP. He added that Claimant's totally disabling pulmonary impairment was due to COPD, chronic bronchitis, and emphysema. These conditions were due to smoking and remodeling secondary to asthma. At his deposition, after reviewing the pathology report of Dr. Bush, Dr. Bellotte opined Claimant

[12] See 65 *Federal Register 245, December 20, 2000, 79941*.

had mild CWP but concluded CWP played no role in causing Claimant's disability. Dr. Bellotte did not discuss whether any of Claimant's emphysema was due to coal mine dust. Moreover, he relied on the pathology report of Dr. Bush whose opinion was discredited for various reasons earlier in this opinion. Perhaps if Dr. Bellotte had access to the report of Dr. Green his opinion regarding causation would have been different.

Lastly, I accord less weight to the highly-qualified opinion of Dr. Fino. He rendered four reports in this case. In his first report he concluded Claimant's disability was due entirely to cigarette smoking. In his second report he agreed that coal mine dust would cause a 2-3 cc drop in FEV-1 and that smoking would cause a 5 cc drop per pack year. Nevertheless he ultimately concluded that coal mine dust made no more than a negligible contribution to Claimant's impairment. By his forth report, he agreed that coal mine dust caused emphysema and that the gold standard for determining the extent of the disease was pathology. He opined that 13 years of coal mining was at the low end of the likelihood of developing significant respiratory impairment due to coal mine dust. He finally admitted that if Claimant did have moderately severe pneumoconiosis as noted by Dr. Green then he could not exclude coal mine dust as being a contributing factor to Claimant's pulmonary disability. It appears that with each new report and as the more information Dr. Fino reviewed in this case he was being backed into a corner where he finally had to admit that he could no longer exclude coal mine dust as a cause of Claimant's disability. Since Dr. Fino essentially changed his opinion with each report, I am not sure what his ultimate conclusion is in this case. Based on the foregoing, I accord the opinion of Dr. Fino less weight.

Accordingly, I find Claimant has established, by the preponderance of the better-reasoned evidence, his total disability was due to coal workers' pneumoconiosis pursuant to § 718.204(c).

Conclusion

Because Claimant has established all elements of entitlement, I must conclude that he has established entitlement to benefits under the Act.

Date of Onset

In a case where evidence does not establish the month of onset, benefits shall be payable beginning with the month during which the claim was filed. 20 C.F.R. §725.303(d). In the instant matter, Claimant filed his claim on June 8, 2000. (DX 1).

Attorney's Fees

No award of attorney's fees for services to the Claimant is made herein since no application has been received. Thirty days are hereby allowed to Claimant's counsel for the submission of such application. His attention is directed to 20 C.F.R. §§ 725.365 and 725.366 of the regulations. A service sheet showing that service has been made upon all parties, including the Claimant, must accompany the application. Parties have ten days following receipt of such

application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

Order

The claim of F.B.W. for black lung benefits under the Act is hereby GRANTED, and

It is hereby ORDERED that ELKAY MINING COMPANY, the Responsible Operator, shall pay to the Claimant, F.B.W., all augmented benefits to which he is entitled under the Act, commencing June 1, 2000.

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MICHAEL P. LESNIAK
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.478 and 725.479. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Avenue, NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).